

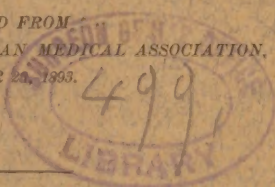
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Hemiparaplegia; with Report of a Case Completely Recovered After One Year's Duration.

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HEMIPARAPLEGIA; WITH REPORT OF A CASE
COMPLETELY RECOVERED AFTER ONE
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In certain lesions of the spinal cord, the prospects of recovery are so much more hopeful from surgical interference than from medical treatment, that an exact diagnosis is of paramount importance. Hence every ray of light, however feeble it may be, that illuminates the question of spinal cord localization, should be most carefully cherished. Our knowledge of the motor centers of the cord is already sufficiently exact to guide the surgeon in his operations, but the precise limitations of the sensory areas are matters still of much uncertainty. A great deal has been accomplished towards increasing our information in this respect by Oppenheim, Westphal, Rosenthal, Eulenberg, Ross, Mills, Osler, Church, and especially Thorburn. According to the general consensus of opinion to-day, the decussations of the sensory and motor tracts are such that a lateral focal lesion anywhere below the cervical enlargement gives rise to a *hemiparaplegia*, with paralysis upon one side of the body and anæsthesia upon the other. Every instance of an anomalous presentation of this classical picture must be possessed of unique interest, hence I beg to present the following case, which has seemed to me to be worthy of special study:

Case.—M. D., a bright, cultured girl 20 years of age and residing in the western part of Pennsylvania, was brought to me Oct. 5, 1890, by Dr. C. J. Steim. The following history was narrated to me by the patient herself in the presence of the

doctor and her elder sister: On the morning of Dec. 25, 1889, while walking along the street, she accidentally stepped upon a loose coal-hole cover and fell in such a manner that the left leg passed down into the coal-chute while the right was extended out over the pavement. She fell heavily with her whole weight. Unconsciousness at once resulted, and on account of the free use of narcotics by her medical attendants, she was not fully cognizant of her surroundings until a week after the accident. In the meanwhile the hip joint was found to have been dislocated and was immediately reduced. There was a considerable hemorrhage from the vagina, and in some way indistinctly described, she was torn. Shortly after the accident the left limb began to swell, became extremely painful and was completely paralyzed. Poultices and hot applications were employed. The fever continued high and with it there was excruciating pain at the back of the head. A week later the swelling of the limb entirely disappeared. Six weeks after the accident, the faradic current was employed locally to the paralyzed muscles without any improvement. In March, three months after the accident, the swelling of the limb reappeared. At this time there was no local sweating, no glossing of the skin, no formation of blisters or sores. This was the last time that the leg swelled. During the course of the year, the right arm enlarged somewhat and became cold and cyanosed. This occurred some five or six times. At no time were there any symptoms of a similar character in the left arm or right leg. There were no cincture pains, no eye disturbances, no dysphagia, no dyspepsia, no dyspnoea. The bowels remained unaffected, with the exception of a few small bleeding piles which appeared immediately after and seemed to be one of the direct results of the fall. The rectal sphincter was very slightly if at all affected. The sphincter of the bladder was much weakened. Severe metrorrhagia occurred some five or six times, and during the entire year there was a profuse leucorrhœa which for a time contained considerable pus. Prior to the accident menstruation had always been painful and scanty, but now had become very much more so. Micturition also produced severe pain. The urine was never voided in normal quantity, but frequently contained a small amount of blood and sediment. At times violent pains shot along the spinal column, especially in the lumbar and lower dorsal regions; while a continuous intense pain was felt in the occipital region of the head. This latter pain was almost constantly present, but on alternate days became so exceedingly unbearable that the patient was obliged to take to her bed and make free use of anodynes and narcotics.

Three weeks before the patient was brought to me, a brilliant stroke of lightning flashed close beside the room

wherein she was seated. She experienced a most peculiar sensation or "kind of rush" up the spine into the head. She fell, became unconscious for a time and continued to be delirious for several days after. For a long while she said she could feel a kind of repetition of the shock about every third or fourth day. Usually the sensation died out in ten or twelve hours. When I saw her she was still subject to intense constricting headaches. These seemed to strike from the back of the head forward to the temples, were always worse in wet weather, and as a rule lasted about twelve hours. Sometimes the face became puffed and pale, while the eyes would be directed inwards. Two weeks after the accident the patient first noticed for herself that her left leg was completely paralyzed and anæsthetic. No improvement had occurred in this respect during the course of the year. So much for the history of the case.

Upon examination I found the eyesight and hearing both good. The grasp of the hands was normal and the same in both. In the right leg the patella reflex, ankle clonus, muscular movements and sensation were all normal. In the left leg they were all completely abolished. The line of beginning anæsthesia corresponded quite sharply with that of Poupart's ligament in front, the crest of the left ilium and a line drawn transversely across the left half of the back on a level with the crest of the ilium or fourth lumbar spine. Above this line there was considerable hyperæsthesia. The limb was well developed, warm to the touch in its upper part, and exhibited the natural color of health. Both the cutaneous and muscular sense were entirely wanting. The deep insertion of a needle into the tissues, the strongest electric currents as well as simple contact with the skin, were absolutely unperceived. So thoroughly did I test for sensation that the possibility of simulation was completely eliminated. A similar examination of the opposite leg and of the two arms revealed nothing abnormal in regard to motion or sensation. Severe pressure along the course of the left crural and sciatic nerves gave no indication of pain. There was no atrophy, as actual measurement showed both limbs to be of the same size. With the use of faradaism, a mild primary current pro-

duced slow but decided muscular contractions, while the secondary current gave rise to more marked muscular movements. The galvanic current produced the usual normal responses. I could detect no indication of the reaction of degeneration. The electrical examination was not, however, as complete as I would like to have made it, as the patient was suffering from considerable pain in the spine and I did not care to distress her further. The distal circulation was slow, and as a result the leg and foot were cold and cyanotic. The whole spinal column was so exceedingly sensitive to the touch that its examination was not so satisfactory as could have been desired. In the region of the lower lumbar vertebræ it was unusually tender, so that the patient winced and screamed with the pain each time I gently pressed the lumbar spines. Between the third and fourth lumbar vertebræ I could distinguish on the left side a small, hard nodule about the size of a pea which was especially painful. The distress and general nervous excitement of the patient prevented my determination at that time of the nature of this swelling. In the vicinity of the sacrum and coccyx the pain upon pressure was quite unbearable.

As a result of this somewhat incomplete examination, I diagnosticated the case as one of incipient meningo-myelitis, with the meningitis as the more pronounced feature and the maximum intensity of the disease focalized on the left side of the lumbar cord. I imagined that at the time of the accident there had been a hemorrhage within the spinal column, either in the lumbar region or elsewhere, and that some of the blood had gravitated and formed an irritative clot which in conjunction with the attending inflammatory process was compressing and constricting certain nerve roots from the left side of the cord. With this conception of the case, I recommended a vigorous daily counter irritation of the entire spine, the administration of mercury (blue mass guarded by opium) to the point of ptyalism, the use of the

mild faradaic current to the paralyzed muscles, and the employment of cod liver oil by local inunction. The treatment was commenced Oct. 5, 1890. About a month later, Nov. 7, or nearly eleven months after the accident and onset of the paralysis, the patient arose from her chair and walked with as much ease apparently as she would have done in perfect health. She stated that a few days prior to my last visit she suddenly felt a kind of tingling and burning sensation, as though the leg had been "asleep," and were recovering, and that this was immediately followed by a complete restoration of its movement and sensation. Hyperæsthesia, I found, had replaced the anæsthesia and though it tired the patient quickly to use the limb its power of motion seemed to have been completely regained. The tingling sensation was noticed at the same time with a similar sensation up and down the entire length of the spinal cord. I found the tenderness of the vertebræ almost entirely gone so that the patient could stand quite vigorous blows upon the back, except in the lumbar region where it was still somewhat sensitive. From this time on she ate and slept better and the bleeding of the hemorrhoids ceased. On Nov. 4, Dr. Steim wrote me as follows: "Patient is steadily improving and is quite as strong as ever she was." On April 28, 1892, I received this note from the Doctor. "I am glad to inform you that Mrs. S. (she had become married by this time) has never had a single symptom of the old trouble since you saw her. In fact her health generally since then has been almost perfect." Again during the summer of last year the Doctor wrote me: "The recovery has been perfect and the patient has been as strong, if not indeed stronger than ever before in her life, with the exception of typhoid pneumonia over a year ago."

The unilateral distribution of the anæsthesia and paralysis in this case indicates, of course, a unilateral lesion. The presence of the motor and sensory paralysis upon the same side would suggest a lesion

outside of the cord proper. The absence of any girdle pain, of pronounced alteration of the sphincter functions, and of atrophy of the muscles establishes the non-involvement of the gray matter of the cord. The upper border of the anæsthetic area limits the upper border of the lesion to the level of the first lumbar segment. The tenderness of the spine, the fever and other constitutional symptoms resulted of course from the meningitis, which was probably a mere extension of the inflammatory process from the lumbar region upwards. The traumatic origin of the paralysis is extremely indicative of a focal hemorrhage and the presence of an irritative clot would be an all-sufficient reason for the inflammation of the membranes. The hemorrhage was probably sub-arachnoid. The transitory symptoms which arose during the course of the year in conjunction with the upper regions of the cord may have been due to a number of smaller hemorrhages of which the blood may have gravitated and so produced a large irritative clot along the line of the lumbar segments and in the meshes of the cauda equina. The chief difficulty in this explanation, however, is the completeness of the anæsthesia from the very beginning of the paralysis; for such a sudden and complete anæsthesia usually follows a lesion in the substance of the cord itself or complete severance of the nerve roots. When nerve roots are compressed by hemorrhagic clots or meningeal adhesions it is more customary for the anæsthesia and paralysis to be preceded by pain, hyperæsthesia, paræsthesia and spasm. Furthermore, with anæsthesia dependent upon destruction of the posterior nerve roots, there is generally very decided atrophy of the corresponding muscles, especially if the lesion involve the root ganglion or that part of the root external to the ganglion.

It must be admitted that there are certain features about this case strongly suggestive of hysterical hemiparaplegia. They are for instance the sex and age of the patient, the location of the paralysis (hys-

terical paralysis being most frequent in the left leg) the absence of any considerable atrophy, the apparent preservation of the rectal with but slight involvement of the genito-urinary functions, the association of the paralysis and anæsthesia in the same area and the peculiar distribution of the anæsthesia which corresponded so closely with Charcot's well-known limitations of anæsthesia in hysterical paraplegia. Against the hysterical hypothesis, however, I place the traumatic origin of the motor and sensory paralysis, the absence of all other hysterical symptoms during this or any previous period of the patient's life, the prolonged duration of the paralysis and anæsthesia without the slightest modification, the absence of the hysterical temperament, the completeness of the anæsthesia and the perfect insensibility of the nerves to the strongest electric currents, the hyperæsthetic zone just above the level of the anæsthetic area and the complete restoration without the slightest subsequent reappearance of any of the old symptoms. Furthermore, the upper limit of the anæsthesia posteriorly corresponded with a horizontal line drawn across the back on a level with the fourth lumbar vertebra, and not with the line of hysterical paraplegia which Charcot says, "follows the insertion of the muscle of the buttocks exclusive of a V-shaped area over the sacrum."

In the *American Journal of the Medical Sciences* for July, 1892 Starr reports a case (No. 5) possessing many points of similarity with our own. Briefly stated, the patient was a woman, 28 years of age, who was well until May, 1889, when after a day of fatigue she was suddenly seized with severe pain in the sacral region and in the back of both thighs, with retention of urine and feces and with a sensation of numbness over the lower sacral region, perineum and vagina. The urine and feces were moved with much difficulty. The sphincter ani contracted on the finger. The knee-jerks were exaggerated. The plantar and gluteal reflexes were normal, and there was no tendency to

bedsores, no tenderness of the back, no girdle sensation nor anæsthesia of the legs. When examined again in Feb. 1890, there was beginning muscular weakness in the left leg and a diminished response to faradaism, without atrophy. There was still some pain in the back and in the left leg constantly but more especially in the left ilio-inguinal region. There was still no girdle sensation, spinal tenderness or bed sore. The area of total anæsthesia had increased. There was slight impairment of the tactile sensation on the outer side of the leg below the knee and on the back of the thigh from the anæsthetic area downwards. The case was not so far advanced as ours, but in the association of its symptoms closely resembled it. The diagnosis of Starr was that of a hemorrhage into the *conus medullaris* and lower sacral segments of the cord. He suggested that this hemorrhage had lighted up a chronic meningo-myelitis and as this advanced slowly upwards, it had increased the symptoms.

Starr and Lloyd's case (No. 11) in the same paper, was one of compression of the *cauda equina* producing a localized paralysis and limited anæsthesia in the right leg. There was a fracture of the lumbar vertebra, followed immediately by a paralysis and indefinite area of anæsthesia in the right leg. A year later there was marked deformity at the third lumbar vertebra, paralysis with atrophy, reaction of degeneration of most of the muscles of the right leg, anæsthesia down the back and outer side of the same limb. In view of the diagnosis of compression of the *cauda equina* at the third lumbar level, Dr. Lloyd operated and removed the second and third lumbar spines and laminae with ultimate recovery of the patient. The point of significance in this case is the association of the anæsthesia and paralysis, which is explained here by the limitation of the lesion to the cauda since the spinal cord proper terminates at the first lumbar vertebra. The absence of the sphincter symptoms would exclude involvement of

the corresponding nerve centers, which Starr finds from two of his cases to be located in the "lower two segments of the cord," a fact which has been confirmed by autopsies in the cases of Kirchhoff, Westphal, Oppenheim and Herter, and in the cases of Rosenthal, Bernhardt, Eulenberg, Mills and Huber. It is always difficult, except in fracture cases, to differentiate lesions of the cauda from those of the cord; and Starr takes the position that it is "questionable whether, except in cases of fracture below the first lumbar vertebra with displacement of the vertebrae any sharp line of distinction between cord and cauda lesions should be attempted." It is to be hoped, however, that some day our knowledge may permit us to make such a differentiation.

In connection with the case I have reported, the question arises as to the possibility of there being a unilateral paralysis and anæsthesia in the same limb, as a result of a single unilateral lesion of the cord. In other words, Is there such a thing as a true hemiparaplegia produced by a focal lesion in which the paralysis and anæsthesia are not crossed? The textbooks almost universally answer this question in the negative. Ranney¹ says: "The muscles below the seat of the lesion are paralyzed on the side of the body corresponding to the exciting cause and the skin is sometimes rendered hyperæsthetic; while the integument of the side opposite the lesion is deprived of sensibility." In his "Lectures on the Nervous System," he furthermore most emphatically states that "should symptoms of anæsthesia appear upon the side where the motor paralysis is present, you may regard it as conclusive evidence that the exciting lesion is progressing and the opposite lateral half of the cord is being involved to a greater or less extent." Seguin² writes that "hemiparaplegia

¹ Applied Anatomy of the Nervous System. Appleton & Co., N. Y. 1888, page 638.

² Pepper's System of Medicine, Vol. v, page 44.

is a rare variety in which one lower extremity is paralyzed while the other is anæsthetic."

Mills³ says that "motor paralysis occurs in the leg of the same side and anæsthesia in the trunk and leg of the opposite side." So authority after authority might be quoted, all stating the same truth. They insist upon the crossed nature of the symptoms. The sensory fibers, after passing through the posterior nerve roots, enter the gray matter of the posterior cornua and at once cross to the opposite side of the cord through the posterior commissure and then continue their course upward to the cerebral cortex. In the upper part of the cord the fibers decussate almost immediately after they enter the posterior cornua but farther down the cord we find them running for a short distance more or less vertically in the side of the cord and in company with the corresponding nerve roots before they pass to the opposite side. In other words, the decussations are relatively higher than the corresponding nerve roots the farther we proceed down the cord. In the words of Gowers,⁴ "The decussation of the sensory tract is not immediate but occurs somewhat above the entrance of the nerves." This, I believe, is of some importance in the diagnosis of our case: for as Gowers again remarks, "A lesion in one side of the lumbar enlargement often affects sensation on the same side as motion because it damages the sensory path before it has crossed." In our case I can not think the anæsthesia was due merely to compression of the nerve roots by the meningeal trouble, because it came on so early and suddenly and was not preceded by the usual hyperæsthesia and initiative signs of local meningitis. In fact, the whole set of meningeal symptoms seems to have been of later development. Nor can I think that the posterior nerve roots supplying the anæsthetic area were them-

³ *Spinal Localization in its Practical Relations*. Detroit, 1889.

⁴ *Diseases of the Nervous System*. P. Blakiston, Son & Co., Philadelphia, 1888, page 166.

selves torn or injured in such a way as to be alone responsible for the loss of sensibility, because the anæsthesia lasted too long to be so quickly recovered from. Torn nerve roots are not usually restored in a few weeks' time by mere external counter irritation and the administration of mercury. I assume, therefore, that the sensory tracts must have been injured somewhere near the periphery of the cord, and that this must have been before the tracts which transmit tactile impressions were separated from those which carry the sensations of pain and temperature since all forms of sensibility were abolished from the anæsthetic area.

According to the most recently constructed tables of segmental spinal localizations, the lesions could not have extended higher than the first lumbar segment. The paralysis and anæsthesia stopped quite abruptly at Poupert's ligament anteriorly and the upper part of the buttock posteriorly. There were no girdle pains whatever, either of the body or of the limb to enable me to determine the level of the lesion more accurately. If the original trouble were a subarachnoid hemorrhage, as I fancy it must have been, the blood may have gravitated to the lower part of the spinal column and involved the fibers of the cauda without doing any special damage to that structure beyond compressing it. A clot may readily have formed on the left side of the cord opposite the lumbo-sacral region, there exerted a pressure sufficiently deleterious to prevent the transmission of the motor and sensory impulses in the corresponding tracts as they passed along near the circumference of the cord, and also to have lighted up a meningitis which gradually extended along the whole column and even implicated to a slight degree the membranes covering the brain especially in the occipital region. Such an explanation is the most reasonable to my mind and harmonizes most satisfactorily some of the discordant symptoms of the case. If such be the correct explanation, the case

indicates that we may have a hemiparaplegia, especially from a lesion in the lower part of the cord, in which the anæsthesia and paralysis appear upon the same side of the body. Further investigations are needed, however, upon this point, though I feel sure that the textbooks are somewhat too dogmatic in asserting that in all cases hemiparaplegia is a paralysis of one-half of the lower part of the body with anæsthesia of the opposite half.

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